ABSTRACT

DEATON, FRAN KIRKSEY. Phase of Alpha Rhythm and Visually Evoked Potentials. (1971)
Directed by: Dr. M. Russell Harter. Pp. 67

The cortical excitability model proposed by Harter (1967) has, in part, been supported by findings in this experiment. The effects of alpha phase, flash intensity, and response task on visually evoked response amplitude, alpha blocking, and reaction time were investigated. The autostimulation technique was used to trigger light flashes, and the "resulting" evoked responses were averaged with a digital computer. Analyses of variance were performed to test for statistical significance on all data from one S. The amplitude of the visually evoked response was functionally related to alpha phase, flash intensity, and the interaction between these two variables ($p < .01$). Intensity significantly influenced early alpha blocking and reaction-time latency ($p < .01$). Two methods of data analysis were used. One method did not take an underlying averaged alpha into account, assuming alpha becomes desynchronized once blocked; the other took averaged alpha into account, assuming that alpha is ever-present but blanketed during photic stimulation. The appropriate model depended upon intensity in this study. High intensity stimulation supported the alpha-desynchronization model; low intensity stimulation supported the ever-present alpha model.

PHASE OF ALPHA RHYTHM AND VISUALLY EVOKED POTENTIALS

by

Fran Kirksey Deaton

A Thesis Submitted to
the Faculty of the Graduate School at
The University of North Carolina at Greensboro
in Partial Fulfillment
of the Requirements for the Degree
Master of Arts

Greensboro, North Carolina

May, 1971

Approved by

M. Russell Harter

Thesis Adviser

APPROVAL

This thesis has been approved by the following committee of the Faculty of the Graduate School at The University of North Carolina at Greensboro.

Thesis Adviser M. Russell Harter

Oral Examination Committee Members Robert H. Eason

Kenneth Smith

May 21, 1971
Date of Examination

ACKNOWLEDGMENTS

The writer's gratitude must first go to her family for understanding the need to carry out this task and to the late Dr. Elizabeth Duffy for encouraging her from the very first.

Dr. M. Russell Harter has been an untiring thesis adviser, and much admiration and gratitude goes to him for his unflagging interest and patient guidance. Appreciation is extended to Drs. Robert G. Eason and Kendon Smith, who served as thesis committee members, for their interest and critical reading of the manuscript.

The involvement and cooperation of the subjects, Lenin Salmon, Gayle Wunder Hock, and Don Hanner, helped to make this study possible.

This research was supported in part by funds received from the National Science Foundation under Grants GB-8053 and GB-7324.

TABLE OF CONTENTS

Table of Contents (continued)

LIST OF TABLES

LIST OF FIGURES

LIST OF EXPERIMENTAL PROCEDURES

Introduction

Numerous investigators have proposed the existence of a cortical excitability cycle (Bergamini & Bergamasco, 1967; Bishop, 1933; Cigánek, 1964; Donchin & Lindsley, 1965; Harter, 1967; Lansing, 1956; Lindsley, 1956) and that the cortical alpha rhythm, recorded from the surface of the scalp, reflects this cycle (Bechtereva & Zontov, 1962; Callaway, 1962; Callaway & Layne, 1964; Callaway & Yeager, 1960; Dustman & Beck, 1964; Lansing, 1956; and Rémond & Lesèvre, 1967). The present study is an investigation of this proposal. If the alpha rhythm reflects the fluctuating excitability of the cortex, it is predicted that there should be a functional relationship between alpha phase at the time of photic stimulation and the visually evoked response (VER), early and late alpha blocking, and reaction-time latency and variability. Prior to discussing procedures and results of this study, research will be reviewed in which the effects of alpha activity and the cortical excitability cycle have been investigated with particular emphasis on the function of the alpha rhythm phase.

The challenge of relating electrical activity of the brain to psychological phenomena has been of interest to many investigators ever since Hans Berger first recorded electrical brain activity in the 1930's. Berger's initial attempts at correlation of neurophysiological and psychological events were not very successful (Andersen & Andersson,

1968; Lindsley, 1952). Nevertheless, the early studies of Berger and others brought forth the clearly observable human alpha and its modification by sensory stimulation.

Alpha Rhythm

Lindsley (1952) considers the alpha rhythm as an abstraction both electroencephalographically and psychologically because it represents an unusual and limited state of affairs in the life of the organism--namely, that of relaxed wakefulness where little sensory information is being processed and attention is wandering.

Lindsley (1952) proposes that alpha activity is a basic metabolic rhythm of the individual brain cell, and its electrical variation alone or in small groups of cells is normally too small to be recorded from the surface of the scalp. He thinks of the alpha rhythm as representing thousands of cells responding in synchrony, which results in sufficient neural summation to produce a recordable alpha rhythm (eight to ten cycles per second, roughly sinusoidal wave) over the posterior head regions in most people (Gaarder, 1966; Kooi & Bagchi, 1964; Lindsley, 1952).

Cortical Excitability Cycle

The idea of a cortical excitability cycle is the synthesis of findings of experiments first started by Bishop (1933) and elaborated by Lindsley (1952). It has remained over the past several decades as a working hypothesis for others (Bergamini & Bergamasco, 1967; Cigánek,

1964; Donchin & Lindsley, 1965; Dustman & Beck, 1964; Harter, 1967; Lansing, 1956; and Rémond & Lesèvre, 1967). It infers that electrical activity of the cortex is able to summate temporally in such a way that there is a period of maximal responsiveness followed by a minimally responsive period. The technique of recording evoked potentials directly from the scalp has made it possible to study this inference directly.

Visual Reaction Time

After the 1930's when Bishop reported stimulation of the optic nerves elicited evoked potentials only in certain phases of the alpha wave in rabbits and cats, Lansing (1957), with the encouragement of Lindsley, continued the investigation of the human alpha rhythm. Following from Lindsley's (1952) definition of the alpha rhythm and the suggestion that it was a good model to represent the waxing and waning of a cortical excitability cycle, Lansing predicted that one or more sequential phases of the alpha wave would correlate with shorter visual reaction times than other phases. It had been noted that simple visual reaction time ranged from 125 to 250 msec. Approximately 50 msec. of this time was thought to be utilized in the transmission of impulses to and from the cortex. The rest of the time was thought to be due to central effects. Lansing surmised that if it could be shown that the alpha rhythm had an excitability cycle in which there were maximal and minimal response periods, it would contribute to

understanding the known variability of reaction time. Lansing studied variations in reaction time in relation to the phase of occipital and motor alpha rhythms in which stimulus and response fell, and he found a functional relationship between alpha phase and reaction time even though, as he pointed out, Walsh and O'Hare (Lansing, 1957) had not found a consistent relationship. Lansing credited his success to using different methods of recording and analysis.

First of all, he pointed out that optimal conditions are necessary for maintaining a uniform state of attention throughout the experiment. His subjects fixated on a red light. Dim visual stimuli were presented 10 to 20 sec. apart in groups of 10 with a rest period between groups. A total of 100 - 200 reactions was obtained for each subject. The alpha phases were designated prior to the experiment, but the flashes were not synchronized to phase. Reaction time to each stimulus was determined for all subjects and later related to phase of the occipital and motor alpha rhythm (measured from brain and tremor rhythms recorded by a Westinghouse Oscillograph) at the time of stimulation and response. Analysis of the data involved the selection and screening of alpha phase with stimuli presentation so that all data used fit the criterion. Finally, only the shortest and longest mean reaction times as they were correlated with phase were statistically analyzed, and they were significantly different ($p < .01$). The computational procedure used was unclear.

Callaway and Yeager (1960) continued the study of visual reaction time and alpha phase but used a different method of triggering the stimuli. Alpha activity recorded from scalp electrodes was amplified and fed into a circuit designed to generate an electrical signal only when phase and amplitude of the alpha rhythm corresponded to a predetermined setting. The electroencephalographic (EEG) activity at the instant of each stimulation was visually monitored and data discarded that failed to coincide with specified requirements. Reaction times were automatically printed out. Callaway and Yeager presented blocks of stimuli at intervals of 10 msec. along 10 phases of the alpha cycle. A comparison was made between the alpha phase at which stimuli elicited the slowest responses and the phase associated with the fastest responses. Their data indicated a more than chance relationship between visual reaction time and alpha phase at stimulation.

Callaway (1962) continued his investigation of factors that influenced the relationship between alpha activity and visual reaction time. Specifically he was interested in the day-to-day variability within the same subject and the effects of stimulus intensity on the relationship between alpha phase and reaction time. His method of triggering stimuli was similar to Callaway and Yeager's (1960). A pencil photocell on the face of an oscilloscope was positioned in such a way that the photocell pulse was generated only when the EEG activity over the range passed by the filter had a specified phase and amplitude.

Reaction times were measured as before. This time subjects, with their eyes closed, were presented three stimulus intensities: (1) strobe for less than 0.1 msec. duration, (2) bright for a 3 msec. square pulse, and (3) dim with a 3 msec. pulse. Intensities were chosen on the basis that they would on the average produce a 50 msec. shift in reaction times. Using vector analysis and 10 phases, Callaway presented evidence to indicate that the alpha phase at which stimulation evokes the slowest reaction time was not significantly or consistently shifted by altering the stimulus intensity. Callaway also concluded that for a given individual there is an enduring tendency for particular phases of the alpha cycle to be associated with fastest or slowest reaction times.

Cortical Evoked Potentials

Meanwhile, the concept of the cortical excitability cycle was being explored by taking amplitude measures of cortical potentials evoked by sensory stimuli that were not time-locked to alpha phase. Research in this area, along with studies concerning the various phases of the alpha cycle, was greatly refined when the technique for averaging evoked potentials was developed. This process involves adding or averaging the values of each co-ordinate of the evoked response over a number of such responses to identical stimuli. Background EEG noise cancels out, and the evoked response, which is time-locked to the stimulus, emerges clearly.

Using these techniques of averaging and recording, many

researchers have studied the evoked cortical potential as it is related to visual and auditory perception (Cigánek, 1961; Donchin & Lindsley, 1966; Gaarder, 1964; Gastaut, Régis, Lijagoubi, & Simon, 1967; Harter & White, 1967, 1968; Katzman, 1964; Kooi & Bagchi, 1964b; Rodin, Gresell, Gudoba, & Zachary, 1965; White & Eason, 1966; Wilkinson, 1967; and Wilkinson & Morlock, 1966). The amplitude of evoked cortical potentials in general not only varies as a function of physical properties of stimuli but also with changes in arousal and attention (Eason, Aiken, White, & Lichtenstein, 1964; Eason, Harter, & White, 1968; Gilden, Vaughan, & Costa, 1965; and Spong, Haider, & Lindsley, 1965).

Others have investigated temporal numerosity and have suggested that the evoked cortical wave form may reflect the periodicity of an excitability cycle with a duration of 100 msec. (Harter & White, 1966; White, 1963).

In any review of the alpha phase research in which the technique of measuring the amplitude of VERs was used, credit must be given to Bechtereva and Zontov (1962), who used a measure of the mean amplitude of the EEG during phase-locked repetitive photic stimulation. They did not have the aid of a signal-averaging computer; even so, they demonstrated that the amplitude of the VER depended upon the phase of the EEG alpha cycle at stimulus presentation.

It was Callaway and Layne (1964) who first used a Computer of

Average Transients (CAT) to study the function of alpha rhythm phase. Visually evoked responses (VER) were obtained from stimulation at four different alpha phases. The effect of alpha phase on the VER was small but seemed to parallel behavioral effects of alpha phase as they were reflected in reaction time.

Dustman and Beck (1964) were impressed with the fact that a cortical excitability cycle was indicated by the relationship between alpha phase and reaction time, but they reported findings had been inconsistent and at times controversial as to the point in the alpha phase which reflected minimal and maximal excitability. They therefore investigated this phenomenon. They used autostimulation for stimulus presentation at various phases, and they used a CAT to record the VERs. In addition to using the CAT, they tape-recorded individual responses so that 10% of all the responses were discarded because they occurred during non-alpha activity. Their RTs were measured from single recordings of muscle (abductor pollicis brevis) activity and converted to time. The slowest 20% of the responses occurring during each phase were discarded to reduce variance caused by inattentiveness. They found savings in RT due to phase to be small but significant ($p < .05$). The largest difference was 6.3 msec. with the slowest and fastest RT and phase correlations being compared.

In addition to reaction-time measurements, a measure of neural activity at the visual cortex coincident with the stimulus-response

sequence was provided by concomitant recordings of the VERs. When they assumed the conduction time from eye to cortex was 57 msec. and corrected for this latency, the fastest mean reaction times were found to fall on the surface negative phase of the wave while the slowest fell on the positive phase.

Rémond and Lesèvre (1967) pointed out that relatively little work had been done in respect to the effects of alpha phase as compared to stimulus and attention parameters in variations in the averaged VER amplitude. Furthermore, the work that had been done showed contradictions. They attributed the conflict not so much to contradictory results as to the fact that measurement procedures were not comparable. In the rare cases in which the amplitude of VER was studied in relation to alpha phase, different components were measured. Dustman and Beck (1965) chose an early wave with a latency of 57 msec.; Callaway and Layne (1964) chose a latency of 136 msec.; and Donchin and Lindsley (1966) chose a later component, 160-200 msec. in latency. Comparable parameters of VER, such as latency, amplitude, or morphology, have not been studied.

Rémond and Lesèvre compared the autostimulation technique (triggering stimuli from alpha phase) to the usual technique of triggering stimuli for the VER. They measured amplitude at component IV (Cigánek, 1961), which has a mean latency of 94 msec., and the earlier negative component III, which has a mean latency of 73 msec.

Subjects, with eyes closed, were stimulated by a stroboscopic lamp for a period of 1 msec. For most subjects, the VER response to autostimulation at the time of maximum alpha source differed from the response to stimulation at a fixed frequency (p=768 msec.) by a slightly increased amplitude and a slightly shorter latency of components III and IV. However, the latency of the components was not significantly affected by alpha phase. Specifically, Rémond and Lesèvre were interested in the effects on VER of four different phases of the alpha rhythm: the time of the maximum of a source and of a sink of the alpha rhythm, and of crossing the baseline before a source and before a sink. The greatest differences were seen when autostimulation was synchronized with the maximum source or sink as measured by presence or absence of rhythmic after-discharge. The after-discharge was not discernible when flashes were given at the time of maximum alpha source and presumably perceived 50 msec. later (due to conduction time lag) at the time of maximum alpha sink; whereas, rhythmic after-discharge appeared greatly enhanced when flashes were given at alpha-sink maximum and presumably perceived at source maximum.

In comparing the averaged alpha rhythm to the early or late rhythmic activity obtained in response to sensory stimulation, Rémond and Lesèvre recognized that these two activities had the same topography and the same frequency but had a considerably greater amplitude. They did not think that early or late rhythmic activity involved the same

neuronal activity as that of spontaneous resting alpha. They concluded that their results suggested that the phase of the alpha rhythm affects the response indirectly by means of a non-specific mechanism of synchronization or recruitment whose release or inhibition transforms the appearance of the VER.

Rhythmic After-discharge

Rhythmic after-discharge, similar to the spontaneous alpha rhythm, has been observed in response to photic stimulation (Cohn, 1964). It was noted that rhythmic 10 Hz waves could be induced by, and were time-locked to, a slowly repeated flash. Study of this phenomenon was greatly facilitated by the advent of averaging techniques and has been the subject of work in the investigation of the physiological basis of alpha activity (Andersen & Andersson, 1968; Barlow & Estrin, 1970). A number of parallels were observed between intrinsic alpha and induced alpha activity. The presence of intrinsic alpha activity appears almost invariably to be a necessary condition for the appearance of the induced rhythmic after-discharge; the frequencies of the two are quite similar, and they both tend to disappear when the eyes are open (Barlow & Estrin, 1970; Cohn, 1964; and Rémond & Lesèvre, 1967).

Rémond and Lesèvre (1967) were able to observe after-activity within a 600 msec. period after stimulation. Goldstein (1970) recorded activity over a 5 sec. interval and, for his screened data, used photic

blocking that was no shorter or longer than 0.5 sec. in duration. Peacock (1970) observed photic blocking and after-discharge for a duration of approximately 2 sec. Neither Goldstein nor Peacock specifically mentioned whether eyes were open during stimulation. Although tracings were recorded for a period of 1036 msec. in the present study, after-discharge was not consistently displayed. This line of research appears particularly related to whether photic stimulation produces a general increase of nonperiodic electrical activity within the occipital region, which might have the effect of overriding an underlying pacemaker, or whether it causes a disruption of cyclical electrical activity (Andersen & Andersson, 1968; Goldstein, 1970).

Two Hypotheses Concerning Alpha Rhythm

Two hypotheses have developed, as a result of empirical evidence, concerning the alpha rhythm. One proposes that the alpha rhythm reflects a cortical scanning mechanism that allows temporal groupings of sensory data into psychological moments (Harter, 1967; Harter & White, 1967; White, 1963). An absolute time base is assumed in which the entire cortex is scanned with all sensory information sampled, coded, and grouped into discrete temporal units. The scanning frequency is assumed to remain fairly constant at the frequency of the alpha rhythm, and the variations in the magnitude and latency of a response to a given stimulus would not be expected within scans (Harter, 1967).

The other hypothesis proposes that the alpha rhythm represents

a cortical excitability cycle which modulates incoming sensory information on a continuous time base. From this hypothesis one would predict that the amplitude of the VER would vary as a function of the temporal relationship between the stimulus and the threshold of the cortical cells. Research specifically designed to study the alpha rhythm as it is related to evoked brain activity indicates that evoked and spontaneous potentials may share a common central neural element (Callaway, 1962; Callaway & Alexander, 1960; Callaway & Layne, 1964; Callaway & Yeager, 1960; Goldstein, 1970; Magnus & Ponsen, 1965; Peacock, 1970; and Rodin et al., 1965).

Harter (1967) proposed a model whereby in the resting or stable state, the alpha rhythm reflects the fluctuating excitability of the system. Whether or not afferent impulses affect the alpha excitability cycle depends on the initiating stimulus intensity and the relative excitation of the cortex. An excitability cycle depends upon the assumption that aggregated neurons are in synchrony, exhibit temporal summation, and have a period of maximal responsiveness followed by a minimally responsive period (Bergamini & Bergamasco, 1967; Cigánek, 1964; Dustman & Beck, 1965; Harter, 1967; Lansing, 1957; Lindsley, 1952; and Rémond & Lesèvre, 1967).

Purpose of the Study

This study extends the work that has been done by others who have investigated the functional relationship of alpha phase at the time

of stimulation and amplitude of the VER and reaction time (Bechtereva & Zontov, 1962; Callaway & Layne, 1964; Dustman & Beck, 1965; and Rémond & Lesèvre, 1967). In addition to studying the effects of alpha phase, three intensities ranging from near absolute threshold to a bright light were used to investigate possible interaction of phase with intensity.

Methodologically, the present study builds on techniques that have been used before in alpha phase research, specifically the use of a triggering mechanism pulsed by the subject's alpha rhythm (auto-stimulation) and a computer to average and record activity during stimulus presentation and alpha activity in the absence of stimulus presentation. Equipment was set up in such a way that the experimenter had only to monitor the polygraph and oscilloscopes during the trial runs, leaving the active participation of the experimenter to the resetting of equipment before sessions and between blocks of trials.

Few quantitative measures have been used in alpha phase research. Dustman and Beck (1965) correlated components of the VER with reaction time. Callaway and Layne (1964) and Rémond and Lesèvre (1967) looked at VER amplitude as it was related to alpha phase, but their measurements were not clearly expressed. In this study, quantitative measures were taken of the VERs from 100-800 msec. after photic stimulation. These measures express changes that can be observed in the VER tracings, specifically the amplitude of the VER component with a mean latency of

212 msec., early photic blocking of alpha, and the possible after-discharge or alpha-like activity after blocking. Assuming that the measures are appropriate, analyses of variance (repeated measures) were used to test for significant differences functionally related to alpha phase and intensity.

Method

Experimental Design

The independent variables in this experiment were alpha phase (with a sixth non-synchronous condition), flash intensity, and the presence or absence of a reaction-time task. The dependent measures were VER amplitude, early and late alpha blocking, and reaction-time latency and variability.

To investigate alpha phase, five equally spaced points in time were selected in the alpha cycle representing Phase 1 at 0 msec.; Phase 2 at 25 msec.; Phase 3 at 50 msec.; Phase 4 at 75 msec.; and Phase 5 at 100 msec. (Figure 1). Phases 1 and 5 were both expected to fall at the trough of the cycle, given the perfectly sinusoidal alpha wave with a frequency of 10 cycles per sec. Over the 180 blocks of trial runs that occurred in this experiment using three subjects (Ss), there were only two Phase 5 block-runs that came questionably close to failing to meet the criterion of $\pm$ 10 msec. in respect to a given phase. Alpha rhythm was averaged to be 10 Hz. In a sixth condition, stimulus presentation was determined by a clock, and the VER was

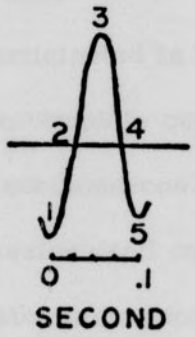

Fig. 1. Phase points expressed in terms of msec. within the 0.1 sec. (100 msec.) alpha cycle at which equipment was set to trigger light flashes.

time-locked to the stimulus presentation only and not to alpha phase.

Three flash intensities were used as photic stimuli. The lowest intensity (0 log units) was near absolute threshold (as discussed below). The other two intensities were two and four log units above this value.

There were two behavioral response conditions. The reaction-time condition (RT) required Ss to respond to a perceived light flash by releasing a microswitch key which the S held down until a flash was perceived. The no-reaction-time condition (NRT) did not require a response at the time of perception.

Each of the three Ss participated in 12 experimental sessions. Six sessions were required to complete one administration of all experimental conditions (all combinations of three flash intensities and two RT conditions). Each session used one flash intensity, and one behavioral response. A session lasted for an hour and consisted of six blocks of trial runs, one for each of the five alpha phases and for the No Synch condition. Each block of trials consisted of 32 presentations of an experimental condition. Ss were permitted to rest between blocks while data were recorded on the X-Y plotter. During the course of the experiment each experimental condition (session) was replicated with the sequence of phase settings reversed. The order in which Ss were subjected to the experimental conditions was counter-balanced within and between sessions and Ss by a Latin square (Appendix B).

Preliminaries

The three Ss chosen for the experiment had been working in the laboratory as graduate assistants and were thoroughly trained. Their ages ranged from 22 to 25 years. Subject LS was adept at producing a good alpha over all sessions. The one female, S GW, was able to train herself to produce an alpha of good amplitude to trigger the experiment; however, in analyzing her data later, it was noted that her alpha activity was quite variable. Subject DH's alpha activity fell somewhere between the other two. His biggest problem was staying alert in a very dark room with little sensory stimulation and no feedback if a flash was missed.

A Ganzfeld mask was made out of a sheet of half-inch pliable foam material. Holes were cut in the eye-socket areas, and a half of a table-tennis ball was glued into each of the holes. The mask was held on the S's head by an elastic which was adjustable to different head sizes. Although it has been a common thought that alpha is blocked when eyes are open, Chapman, Shelburne, and Bragdon (1970), Mulholland and Evans (1965 and 1966), and White and Eason (1966) are of the opinion that alpha is blocked because of contours that come into the field of vision. The Ganzfeld was used to reduce marked contours and to diffuse the light; with it, Ss did in fact show alpha rhythms with their eyes open.

Ss were able to practice producing their alpha rhythm. Whether or not a light was flashed depended upon alpha activity of a certain amplitude, which pulsed a Lehigh Valley electronic (LVE) adjustable

Schmitt Trigger that started a trial run. Subjects were told that trials could be as close as four seconds apart (the minimal between-stimulus interval) and that if they were less often, it was because sufficient alpha activity was not present. Actually the delay in feedback (100-200 msec.) by a flashing light was not ideal for alpha conditioning, but even so, it proved helpful.

While Ss were practicing in the experimental setting, the lowest flash intensity was adjusted to the point where S DH was able to perceive it only around 50% of the time. The other two Ss perceived it on the average of 90% of the time. This intensity was used as basic for each of the three Ss. The intensity of the flash was varied by removing neutral density (ND) filters situated between Ss' eyes and the light source (a 10 μsec. flash produced by a Grass S-2 Photo-Stimulator set on the intensity level 2). A 2.0 log ND filter was removed for the medium intensity, and a 4.0 log ND filter for the highest intensity. The duration of the flash remained constant for all sessions.

The microswitch used to measure reaction time was familiar to Ss, and they had no trouble releasing it with a minimum of muscle involvement. In order to have a record of the number of flashes perceived during the NRT condition, Ss were asked to push a button (which activated an event mark on the polygraph record) with their left index finger, after they heard a click which was programmed to be sounded over the white-noise system 1036 msec. after the beginning of the trial run. They were

to push the button in response to the click if they had not seen a flash. This same click was used to give feedback to Ss as to RT latency in the RT conditions. The click was presented when Ss responded too slowly and indicated the end of a trial run.

Procedure

The S, stimulus display, and two devices for signaling responses were located in a light- and sound-shielded room. The remainder of the equipment was located in an adjoining experimental area. An intercommunications system was on at all times. White noise, generated by a Model 901B Grason Stadler noise generator, was used to mask extraneous noises during the trial runs.

Before each session, the S put on dark-adaption goggles, and he was prepared for the experiment. Electrode jelly was rubbed into the scalp 2.5 mm. above the inion on the midline and on the right ear lobe. A gold-plated 8 mm. electrode was secured at the scalp area, and the reference electrode was clipped on the ear. Skin resistance was reduced below 10,000 ohms. The electrode leads were connected to an EEG input terminal located beside the chair, and this in turn was connected to the polygraph in the adjoining room.

The S was placed in a comfortable chair with head and arm rests that helped him maintain a relaxed upright position. He was advised to keep his chin tucked in order to minimize muscle tension in the dorsal neck muscles. The RT key was on the right arm rest. The report button

was on the left arm rest.

The stimulus display was 80 cm. in front of the S at face level. A 17x17 cm. translucent screen was mounted on the end of a 47.5x57.5x100 cm. cardboard box. The stroboscopic lamp (strobe) was mounted on the other end of the box and was easily removed for placement of the ND filters. Actually then, the S was seated 180 cm. from the source of light which flashed through the translucent screen and was to be perceived as a Ganzfeld.

Instructions were given to the S concerning stimulus intensity and the response required. He was reminded that his alpha rhythm triggered the trial run which was programmed for a minimum interval of four sec. He was told that approximately 32 light flashes would be presented randomly in the run of 64 trials, so that 50% of the time there would not be a flash. The room was darkened; the dark-adapting glasses were removed; the Ganzfeld mask was placed in position; and the E left the room. The S was asked to relax but to try to see the flash while the equipment was being adjusted.

Apparatus

Electroencephalographic activity was preamplified by two Grass Model 7P5A Wide Band AC EEG filters and amplified by two Grass Model 7DAC DC Driver Amplifiers. Activity connected to the LVE Adjustable Schmitt Trigger (for autostimulation) was highly filtered with half-amplitude high- and low-frequency filters set on 15 and 3 Hz, so that

10 Hz activity would be the primary activity passed. The other polygraph channel was used to filter EEG activity feeding into the computer for averaging. The half-amplitude settings were 35 and 1 Hz.

As was mentioned earlier, this experiment depended upon the presence of alpha activity of a certain voltage to pulse a LVE Adjustable Schmitt Trigger (ST). The setting for the ST was adjusted for each S. During the first six sessions the criterion for amplitude was lower than that for the last six sessions. The first six sessions for S LS and S DH required a voltage of 8 μV to pulse the trigger. The last six sessions for Ss LS and DH required a voltage of 36 μV and 18 μV respectively. A 36 μV wave was required to activate the ST throughout the experiment for S GW.

Once the trigger had been activated, the trial run was in control of and programmed by the solid-state equipment, most of which was Lehigh Valley electronic modules. The ST activated a recycling time set at 4 sec. so that only one trial run was possible during that interval. The ST simultaneously started another recycling timer set at 25 msec. which, in conjunction with a binary predetermining counter, determined the five phases (0-100 msec.). The appropriate phase was set before each trial-block. The digital computer and the monitoring oscilloscope were set to record a 1036 msec. epoch. A probability gate was also activated which was set at 50%. During the course of a trial-block, 32 $\pm$ 2 photic stimuli were flashed randomly throughout a total of

64 activations of the Schmitt Trigger. The flash came from a Grass Model PS-2 Photo Stimulator set at intensity #2 after a 100 msec. delay. This 100 msec. delay enabled a complete alpha cycle to be recorded in the computer before stimulus presentation. The amplitude of this cycle was later used to determine the baseline for measurement of the VER amplitude. During the RT conditions the Hewlett Packard electronic counter was turned on to print out reaction times.

Mention must be made of the fact that there were not always 32 light flashes presented in each block of 64. Due to limitations of the programming equipment, 32 ± 2 light flashes were actually presented. Thus, both stimulus and non-stimulus presentation trials amounted to 32 ± 2.

During the trial runs the Hewlett Packard 141A Oscilloscope permitted the experimenter to monitor the on-going EEG activity and time of stimulus presentation. This arrangement provided moments of both joy and frustration. Perfect alpha activity (as was the case 90% of the time) drew applause; muscle or other artifacts drew groans. The bad sweeps or samples just had to be tolerated with hopes that they would not influence VERs too adversely.

In addition to starting and programming the equipment used in presenting the random light flashes and recording the reaction times, the solid-state system relayed all data to a Fabri-Tek Model 1062 Instrument Computer with Model SD-2/4 Four Channel Signal Digitizer and

SW-2 Sweep Control Plug-Ins. The EEGs were recorded monopolarly from the S, filtered by the polygraph as mentioned earlier, and averaged by the computer over the trial-block of 64 trial runs, 32 ± 2 randomly presented stimulus (SN) and no-stimulus (N) trials. In the autostimulation conditions, it was assumed that both the alpha rhythm (noise or N) and evoked response (signal or SN) were present to some degree. Thus these are termed "SN" conditions. Trials when the stimulus was withheld and only the alpha rhythm averaged were accordingly termed "N" conditions. The No Synch trials were activated by a clock instead of the ST, the resultant being a typical VER.

Averaged records (Figure 2) obtained during the SN condition were stored in the first computer register and the N conditions in the third register. The second register recorded the time of stimulus presentation and the reaction-time histogram (RT condition). The fourth register was used to subtract N from SN conditions. Data from this register were not used for quantification and analysis because of the unequal summations.

The summated VERs were made visible by a Tektronix Type RM 504 Oscilloscope. A Hewlett Packard 7035 X-Y Recorder plotted out the data that were later used for amplitude measures. The X-Y Recorder was calibrated to display 5 μV per half-inch with a summation of 64. Except for the No Synch condition, Figure 2 is an example of a typical record of averaged activity obtained under one condition (Phase 5, high inten-

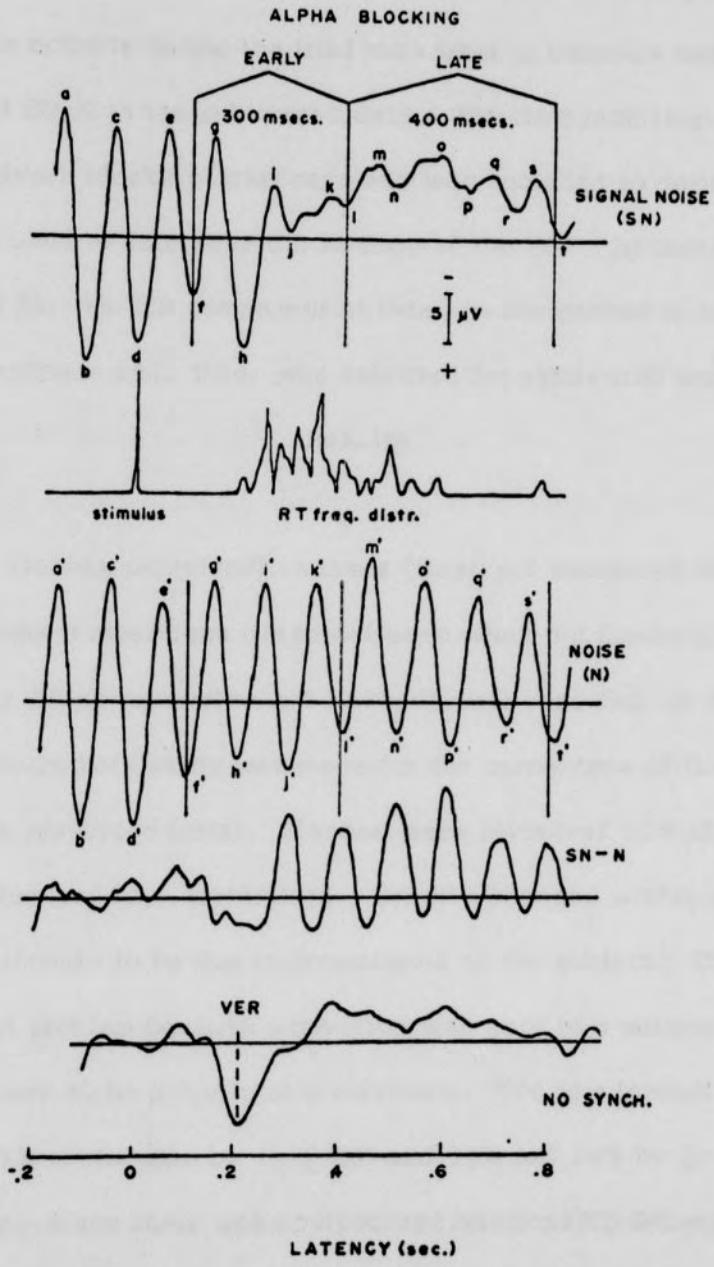

Fig. 2. Typical trial run from which all amplitude measures of VER, early and late alpha blocking, and average alpha were taken.

sity, RT): SN contains averaged activity when signal was presented; N is the alpha activity during the trial runs when a stimulus was not presented; and SN-N is the subtracted data. The No Synch is a summation of two No Synch blocks of trial runs and was included to demonstrate how it was used to determine the latency of the VER. In terms of all the data for all Ss, the VER component at this latency proved to have the greatest amplitude and, thus, was selected for statistical analysis.

Results

Hits

Hits (flashes perceived), misses (flash not perceived when flashed), correct rejections (flash not seen when not flashed), and false alarms (flash seen when not flashed) were recorded on the polygraph. A preliminary study was made for the percentage of flashes perceived when presented (hits). Flashes were perceived 92% of the time for the medium and high intensities. Drastic changes within a block of trials were thought to be due to drowziness of the subject. This was an ever-present problem because activation was kept to a minimum in an attempt to keep alpha activity at a maximum. The low intensity was perceived 47% of the time by S DH and 94% and 84% by Ss LS and GW respectively. Since there was no apparent relationship between alpha phase and percentage detection, no further analysis of these data was made.

Visually Evoked Responses

Superimposed tracings were made for all Ss over all sessions. Figure 3 shows the effects of alpha phase on data from three Ss (LS, GW, and DH), under the RT condition, when the 2 log unit stimulus was presented at the various alpha phases (SN), when the stimulus was withheld and the alpha rhythm averaged (alpha or N condition), and when the stimulus was presented randomly in respect to alpha phase (No Synch or VER condition). Figure 4 shows similar data except the effects of three flash intensities are shown for one S (LS) under the NRT condition. Each tracing represents the summation of 32 $\pm$ 2 responses. It should be remembered that the SN and N conditions were presented randomly while viewing these figures.

Visual inspection of the superimposed tracings from all Ss revealed that the first evoked potential component that changed appreciably from that of averaged alpha activity was surface positive, occurring at a mean latency of 212 msec. Latency of this VER component was determined for each subject by averaging the latency of all 12 of the No Synch tracings. Latencies for Ss LS, GW, and DH were 217 msec., 208 msec., and 210 msec. respectively. Subject LS's VER(N) amplitude in response to the 2 log unit (medium) intensity shows a sudden decrease at Phase 2 in comparison to Phase 1. Then the amplitude of the VER(SN) can be seen to gradually increase until reaching Phases 4 and 5. In looking at his tracings alone (Figure 4) it can be seen that VER(SN) amplitude also varies as a function of intensity. The tracings in Figure 3 of the

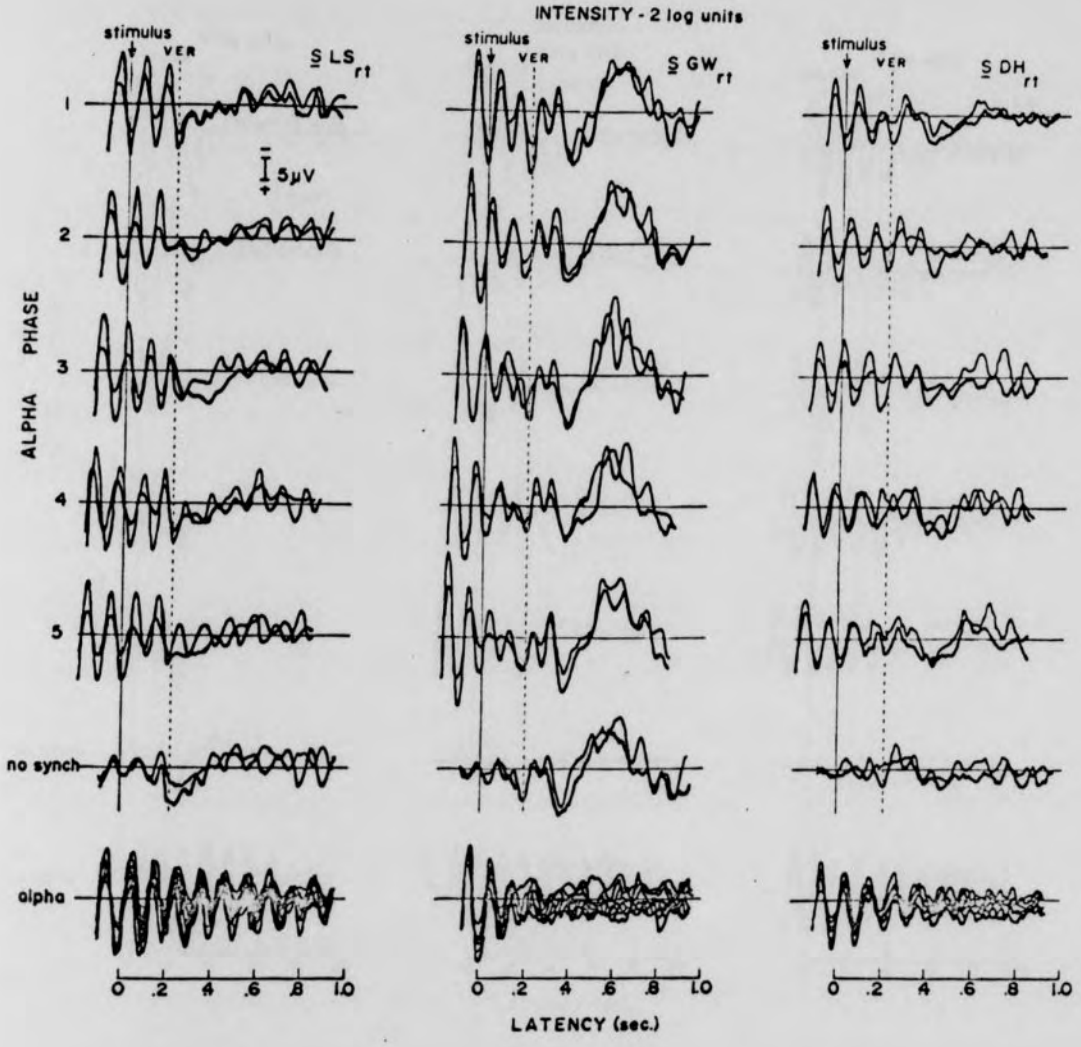

Fig. 3. Effects of alpha phase at 2 log units above threshold on averaged activity with autostimulation obtained from Ss LS, GW, and DH. RT conditions only. Each tracing represents the summation of 32 ± 2 responses.

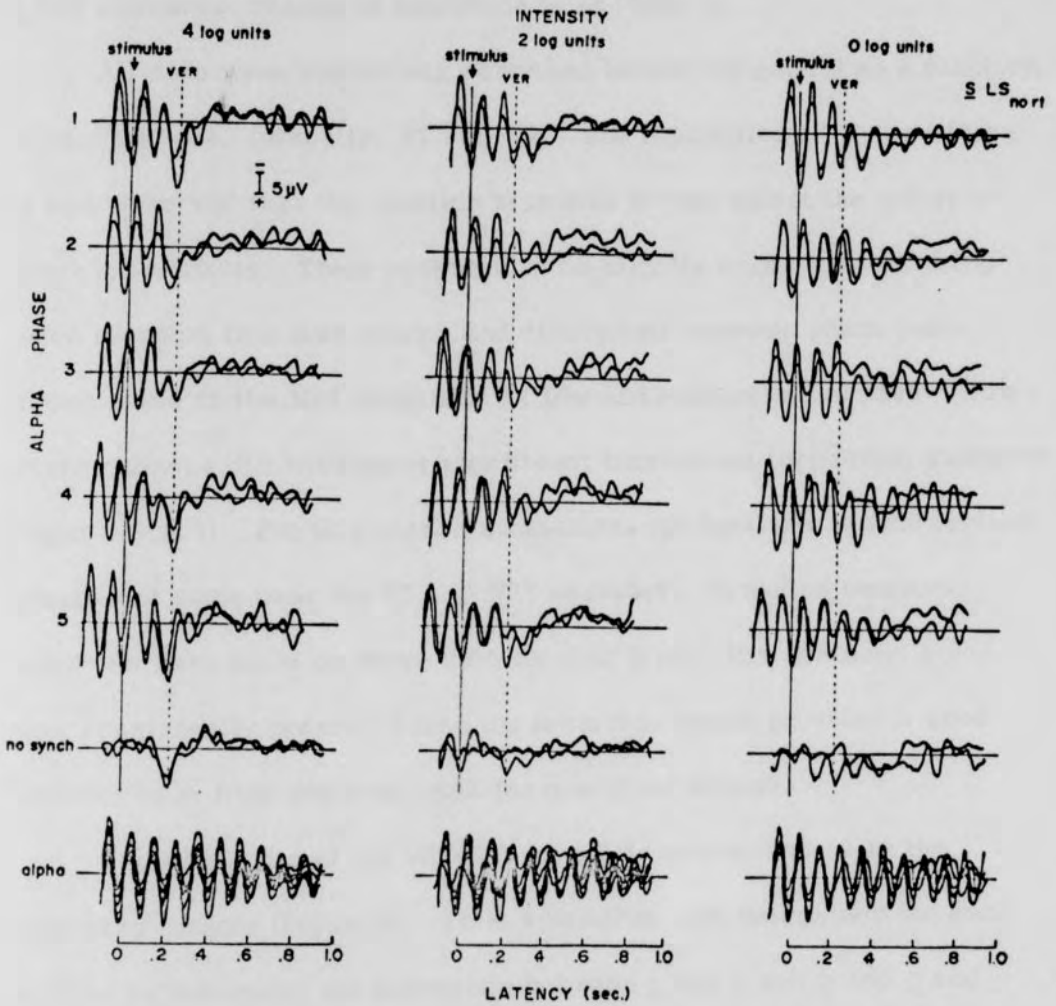

Fig. 4. Effects of alpha phase and 3 flash intensities (log units above threshold) on averaged activity with autostimulation obtained from S LS. NRT conditions only. Each tracing represents the summation of 32 ± 2 responses.

VER(SN) for Ss GW and DH also display changes. Subject GW has the same sudden change in amplitude at Phase 2 as does S LS: whereas S DH's greatest change in amplitude is at Phase 4.

All data were plotted (as described below) for each S as a function of alpha phase, intensity, RT and NRT, and replications. By and large it was observed that the reaction response did not affect the nature of averaged activity. There appeared to be slightly more alpha blocking when reaction time was taken, and differences between phase were accentuated in the NRT condition for low and medium intensities. However, changes did not appear significant from visual inspection (compare Figures 3 & 4). For this reason summations for first and second replications were made over the RT and NRT sessions. Repeated measure analyses were made on these data for only S LS. His averaged alpha was consistently present during all sessions, which provided a good working base from which to work (as described below).

The amplitude of the VER(SN) for all Ss was measured in the following manner (Figure 2). First a baseline was determined for each tracing by averaging the difference between a and b and b and c and dividing by 2, giving the theoretical 0 point from which deflections are surface negative (up) or surface positive (down). Response amplitude was measured vertically in reference to this baseline. As has been mentioned previously, an individual latency was determined for each S. The results of the VER(SN) measurements were plotted as a function of

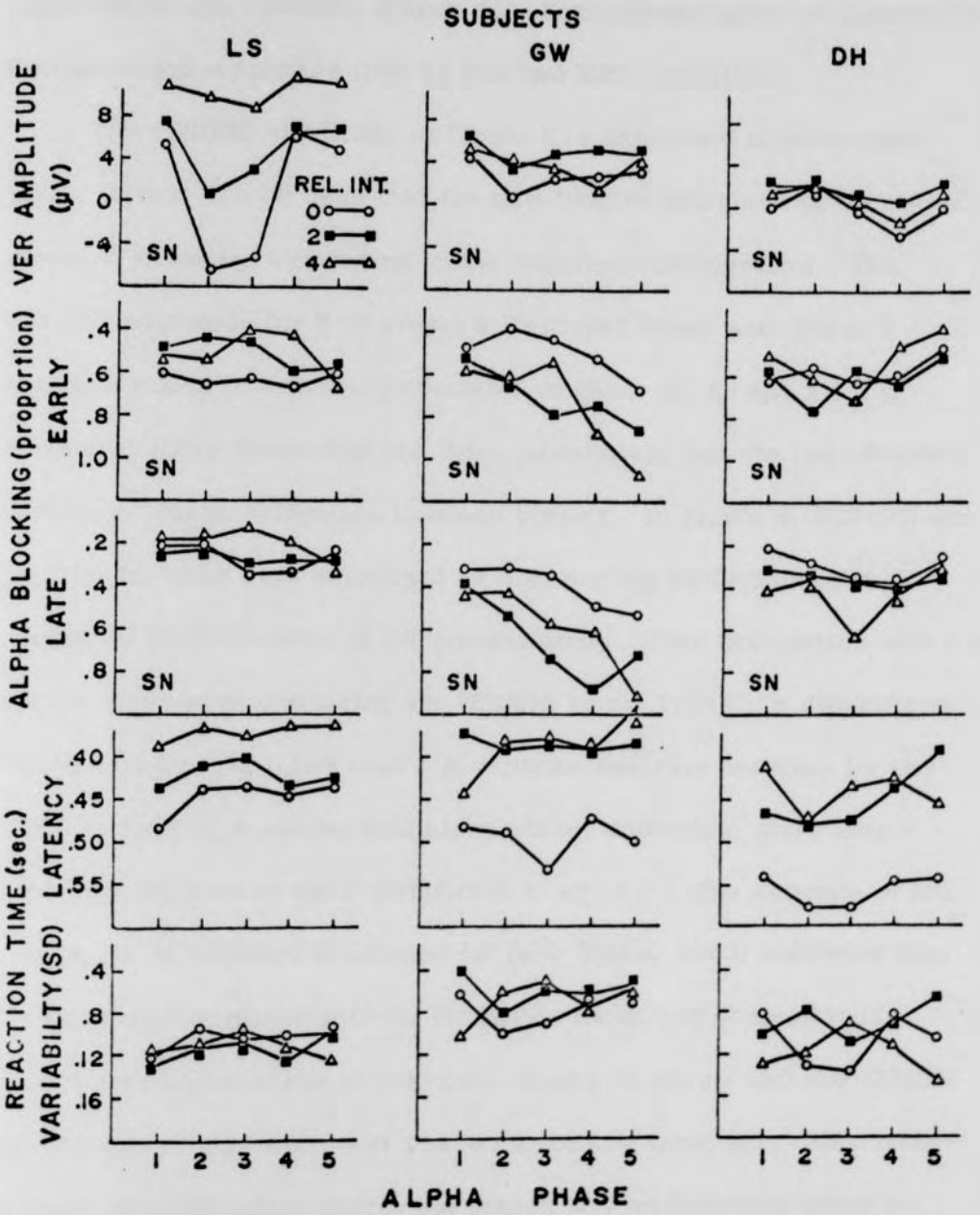

Fig. 5. Dependent measures for Ss LS, GW, and DH under auto-stimulation.

alpha phase and intensity (Figure 5). Each plotted point is the mean of four averaged responses (two RT and two NRT conditions).

The VER(SN) amplitude in Figure 5 is expressed in microvolts (μV). Here it can be seen that the quantitative measures taken are expressive of changes observed in the superimposed tracings. The VER(SN) amplitude for S LS shows a U-shaped curve with Phase 2 having a negative amplitude compared to Phase 1, 4, and 5. The U-shaped curve holds over the three intensities, but the low intensity reveals a greater difference between phases. In Figure 6, VER(SN) for S LS is the same data expressed as a proportion by dividing the μV amplitude by the number of SN presentations. This calculation was done for the purpose of comparing the VER(SN) to the VER(N), a comparison which will be discussed later. A variance analysis summary for the VER(SN) for S LS revealed that alpha phase, intensity, and Phase X Intensity interaction were significant ($p < .01$). The estimate of the proportion of variance accounted for (w^2) (Hays, 1963) indicated that these three significant factors accounted for 61% of the variance. Visual inspection of the plotted data (Figure 5) shows that the VER(SN) amplitudes change more over phase for the low intensity. This interaction was anticipated and is the reason why an intensity close to threshold was chosen.

Plotted data of the VER(SN) amplitude (Figure 5) for Ss GW and DH also show changes, but to a lesser degree between phases. The

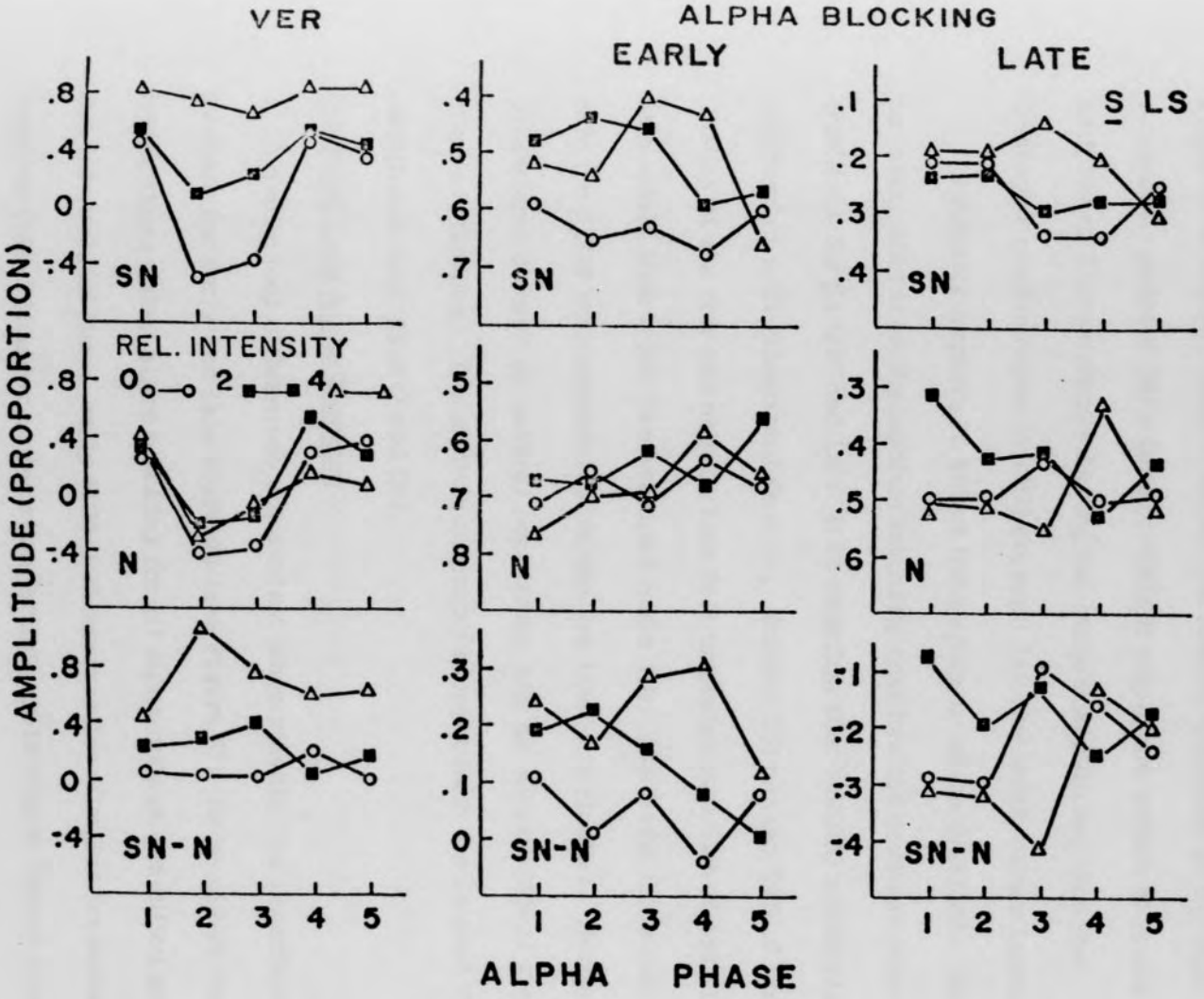

Fig. 6. Amplitude of cortical potentials for S LS expressed in proportions.

skewed U-shaped curve is present in the plotted data for S GW over the medium and low intensities, with the lowest amplitude being at Phase 2; however, Phase 4 shows the lowest amplitude at the high intensity. Subject DH's lowest VER(SN) amplitude occurs at Phase 4, with Phase 2 consistently having the highest amplitude; thus the U-shaped configuration is still present, but the break occurs later.

Intensity appears to affect the amplitude of the (VER(SN). Note, however, that it is the medium intensity that has the greatest over-all amplitude for Ss GW and DH. It is possible that VER(SN) amplitude is confounded with attentional factors. Subject DH missed 53% of his flashes at the low intensity. This fact indicates that 53% of his SN data would look a lot like averaged alpha (N). The same thing can be said for S GW who missed 16% of the low intensity flashes. Subject DH became drowsy on several occasions, and he missed 15% of the brightest flashes. The medium intensity brought forth the largest VER amplitudes from Ss GW and DH.

Early and Late Alpha Blocking

Individual differences are evident when viewing the superimposed tracings for early and late blocking (see Figure 3). As an aid in analyzing these data, alpha blocking for all subjects was quantified over two intervals of time: early (100-400 msec. after stimulus presentation) and late (400-800 msec. after stimulation). Referring to Figure 2 data from early alpha blocking (AB(SN)) included measures f-g, g-h, h-i,

i-j, j-k, and k-l. These measures were summed, divided by six, and divided by the average of c-d and d-e. The measure expresses alpha blocking in proportion to the amplitude of averaged activity at the time of stimulus presentation and can be compared to any other SN or N data obtained in the same manner, regardless of an unequal number of summations. Late alpha blocking was analyzed in the same manner, with the peak to trough measurements going from l to t. The results were plotted as a function of alpha phase and intensity (Figure 5). Each plotted point is the mean of four measures (two RT and two NRT conditions).

Visual inspection of Figure 5 and the early AB(SN) and late AB(SN) data reveals phase to be of questionable significance for Ss LS and DH; however, phase does appear to be significantly different in plotted data for S GW, with Phases 1 and 2 having less alpha-like activity than Phase 5. Phases 1 and 5 are supposed to be essentially identical within the 100 msec. alpha cycle. These data, therefore, reflect the rapid attenuation of the averaged alpha that was observed in the raw data, and not the effects of phase. Intensity appears to be functionally related to the extent of alpha blocking for all Ss; however, there is inter-subject variability. Plotted data for S LS indicates that less alpha blocking occurs at the low intensity and the most at the high intensity; whereas plotted data for S GW reveal that the most alpha blocking occurs at the low intensity. Intensity appears to have little

effect on alpha blocking for S DH.

The variance analysis summary for AB(SN) for S LS (Tables 3 & 4) revealed that the effects of phase on early or late alpha blocking did not approach statistical significance ($p > .05$). Phase X Intensity interactions did not approach significance ($p > .05$), but accounted for 13% of the variance in the early date. Phase X Replication interactions were also non-significant ($p > .05$) even though they accounted for 9% and 13% of the variance for the early and late AB(SN) data, respectively. Intensity had a significant effect on early AB(SN) ($p < .01$) and accounted for 28% of the variance. The effect of intensity was not significant for late AB(SN) ($p > .05$), accounting for only 12% of the variance.

Reaction-Time Latency and Variability

Reaction-time latency and variability were calculated for all Ss by using the print-out data from the electronic counter and printer. The results were plotted as a function of alpha phase and intensity (Figure 5). Each plotted point is the average of two replications.

Visual inspection of reaction-time latency suggests that RT latency is functionally related to intensity, with fastest RTs occurring at the high intensity. Phase is not significant ($p > .05$) in the analysis of variance for S LS. Intensity is significant ($p < .01$). See Table 4. Since the analyzed RT findings do not go along with what has been found previously in other research, mention will be made of this in the

discussion section of this paper.

Reaction-time variability for S LS was variable, showing no functional relationship with phase or intensity. This was demonstrated by the analysis of variance on these data from S LS (see Table 5).

In addition to the above mentioned measurements for the three Ss, additional measurements were made on all the data from S LS. His data were selected in particular because his averaged alpha was so consistent during the no-signal trials (N) (Figures 3 & 4). The original VER(SN) μV amplitude measure was divided by the number of summations. A corresponding measurement was made on the averaged alpha or VER(N). Corresponding early and late alpha blocking measures were made on the averaged alpha activity (early AB(N) and late AB(N), respectively) as were made for SN. Specifically, referring to Figure 2, measurements were made from f'-l' and from l'-t'. Since all amplitudes were expressed as proportions, it was then appropriate to subtract VER(N) from VER(SN). The results were plotted as a function of alpha phase and intensity (Figure 6). Each plotted point is the mean of four measures.

Research specifically designed to study the alpha rhythm as it is related to evoked brain activity indicates that evoked and spontaneous potentials may share a common central neural element. For this reason, analyses of variance (repeated measures) were made for VER (SN-N) and early AB(SN-N) and late AB(SN-N) (see Tables 6, 7, & 8).

Visually Evoked Responses Minus Averaged Alpha

Variance analysis summary for VER(SN-N) revealed phase to be significant ($p < .05$), as was intensity ($p < .01$). The Phase X Intensity interaction did not approach statistical significance ($p > .05$). A total of 84% of the variance was accounted for, 72% being due to intensity.

Early and Late Alpha Blocking Minus Averaged Alpha

Phase did not prove to have a significant effect on early or late alpha blocking (SN-N). Intensity for early alpha blocking (SN-N) was significant ($p < .05$) with a w^2 of 19%. Intensity was not significant for late alpha blocking (SN-N) but accounted for 16% of the variance (Tables 7 & 8).

Summary of Analyses of Variance

Intensity significantly influenced all dependent measures except RT variability, and late AB(SN) and AB(SN-N) and accounted for most of the variance. Changes as a function of alpha phase were significant for both VER(SN) and VER(SN-N) amplitude measures; however, variance accounted for was minimal compared to that of intensity. More variance was accounted for with Phase X Intensity and Phase X Replication interactions. Examination of Figure 6 indicates that Phase 1, 4, and 5, as drawn in Figure 1, are the most excitable. Actually these three phases are within 25 msec. of each other, with Phase 1 and 5 being ideally the same point in time within the alpha cycle (100 msec.). Phases 2 and 3, in comparison, are the less excitable and occur from 25-50 msec.

later in time from Phase 1, 4, and 5.

Although effects due to replications are not great, they do appear to approach significance as interactions in all the data analyses except for early and late alpha blocking (SN-N) and RT latency. This observation indicates that there is some day-to-day variability, which agrees with findings of Callaway (1962), Magnus and Ponsen (1965), and Bechtereva and Zontov (1962). In the case of early and late alpha blocking (SN), replications accounted for 11% and 23% of the variance as interactions.

Discussion

Reaction time was one of the first psychological phenomena found to be related to alpha phase. It was anticipated that this experiment would also find a significant relationship, but this did not turn out to be the case. What are the reasons?

First of all, all the data were recorded and averaged. Averaged alpha was used instead of the individual alpha and stimulus presentation tracings; so that more random non-alpha activity was involved than in the other experiments mentioned. There was considerable variability in the RTs. As Lansing (1957) mentioned, variations in set and attention critically affect RT. He used short trial runs with rest periods between runs. He also used a fixation light. Subjects in the present experiment, sitting in a totally dark room and responding to a Ganzfeld, had difficulty even knowing whether or not their eyes were

open (they were supposed to be) or rolled up into their head. Also, all RTs were used (in one S) and all phases analyzed in one repeated measures design. In that analysis, phase did not prove to be significant. After going back over the data and comparing mean RTs between Phases 1 and 3, however, there was a 46 msec. difference with the low intensity, 37 msec. with the medium, and 11 msec. difference with the high intensity. Phase 3 correlated with the shortest mean RT. In looking at Figure 5 a trend of this kind can be seen with S LS: there is a reverse in trend for Ss GW and DH, showing Phase 3 to be correlated with the longest RTs. Phase 3 in this experiment is the peak of the negative deflection in the 100 msec. alpha cycle; no corrections have been made for transmission time to the cortex.

The alpha average was chosen to work with because of its relatively simple characteristics and the ease by which it is derived. Precisely, alpha average is averaged activity correlated with the alpha rhythm through the period explored. Within the same subject, averaged alpha frequency is extremely stable (Rémond, Lesèvre, Joseph, Rieger & Lairy, 1969). In the case of S LS, the fact that his averaged alpha attenuated slowly indicates his spontaneous alpha rhythm has a particularly restricted frequency band; whereas S GW whose averaged alpha attenuated rapidly indicates that her alpha rhythm has a more widely dispersed frequency. It is evident that the more stable and free from noise the spontaneous alpha rhythm is, the more the alpha average

obtained will be reproducible and representative of the spontaneous alpha rhythm. In the present study, as has been mentioned earlier, the criterion for stimulus presentation at a particular phase $\pm$ 10 msec., was met in the 180 trial runs performed (Figures 3 & 4). Even though using averaged alpha is a less precise procedure than using true correlations, it has the advantage of being faster to perform. (See Rémond, Lesèvre, Joseph, Rieger, & Lairy, 1969; and Joseph, Rémond, Rieger, & Lesèvre, 1969 for a thorough description of the alpha average.)

The use of the averaged signal activity (SN) proved to be detrimental to the study of the alpha-phase relationship to reaction time; however, it proved to be very helpful in obtaining the measurements for the VERs and early and late blocking. In addition, it was possible to average concurrent alpha activity during the no-stimulus condition so that there would be some idea of what type of brain activity occurred when a stimulus was not presented. That is to say, this information gives some notion of how long averaged alpha lasts, whether or not it attenuates, and if so, in what way.

There is no way that this experiment can settle the question of whether or not a photic block calls forth participation of additional neurons, but it can be said that measurement of the VER across intensities shows that the VER(SN) at the low intensity does not look to be appreciably different from the averaged alpha VER(N) at the same point

in time in spite of the fact that 94% of the time a flash was perceived (see Figure 4 & 6); whereas VER(SN) amplitude at high intensity no longer resembles that of the averaged alpha. This observation could mean either that alpha has become completely desynchronized or that a general increase in nonperiodic electrical activity within the occipital region might have the effect of overriding an underlying pacemaker (Goldstein 1970). Goldstein held that, if there were an underlying pacemaker, the temporal phase relationship between pre- and post-blocked sections of a given alpha wave train would be unaffected by the onset or sessation of a photic stimulus. His research supported the paced generator model. As was the case in the successful reaction-time experiments, Goldstein was in the position to screen out muscle potential artifacts or violent spontaneous phase discontinuities. In addition, onset of the light pulse was not synchronized with the alpha wave. The correlation data were selected later. Here again, using the averaged VER and alpha technique may have been a too insensitive procedure to be sure about temporal phase continuity. With the data that were secured in this experiment, however, and with what little afterdischarge that occurred that looked as if it could be called such, it appeared as if there had been a phase shift between the pre- and post-blocked averaged alpha wave.

Supposing, however, that there is an underlying pacemaker that continues to perform during photic blocking, then perhaps, it would be

fitting to take the averaged alpha that was randomly secured during each block of trials and subtract it from the stimulus trials. This was done in the SN-N analysis of variance. Phase significance dropped from $p < .01$ to $p < .05$ and Phase X Intensity interaction dropped in significance from $p < .01$ to $p < .10$; however intensity did not change from the $p < .01$ level. As a matter of interest, 62% of the variance was accounted for in the SN data; whereas 84% of the variance was accounted for in the SN-N data, 72% being due to intensity (Tables 1 & 6).

Alpha phase at the time of stimulation did not appear to be functionally related to early and late blocking (SN-N) at least to the extent that there were no significant differences in the amount of blocking from one phase to another. The difference observed (Figures 4 & 6) in alpha blocking was due to the intensity of light flash. Subtraction of the averaged alpha left intensity to still be significant at the $p < .01$ level; however, the amount of variance accounted for by intensity dropped from 28% to 19% (Tables 4 & 7).

Late alpha blocking was even less sensitive to alpha phase and intensities, and the after-discharge that others have gotten under comparable experimental conditions (Rémond & Lesèvre 1967); Horstfehr 1967; Goldstein 1970) was not consistently present. It has been mentioned that after-discharge does not occur with opened eyes. No information was given concerning the visual field when eyes were opened. For the interval of time between 400 and 800 msec. after

stimulus presentation, none of the independent variables used had any significant functional relationships (Tables 3 & 8).

Summary

The cortical excitability model proposed by Harter (1967) has, in part, been supported by findings in this experiment. The effects of alpha phase, flash intensity, and response task on VER amplitude, alpha blocking, and RT were investigated.

The autostimulation technique was used to trigger light flashes, and the "resulting" evoked responses were averaged with a digital computer. Data which were recorded by an X-Y plotter were analyzed by averaging over four measures (2 RT and 2 NRT conditions) and plotting for all Ss. Analyses of variance were performed to test for statistical significance on all data from one S.

The amplitude of the VER was functionally related to alpha phase and intensity; there was also an interaction between the two, there being a greater difference due to phase at the low intensity. Intensity significantly influenced all dependent measures except for RT variability and late alpha blocking and accounted for most of the variance.

Discrepancies from previous research in the RT data are thought to be due to differences in methodology, technically and statistically.

No attempt was made to determine the physiological basis of alpha nor the point of hyper- or hypo-excitability of the alpha cycle at the cortex. Two methods of data analysis were used. One method used

only the SN data and did not take an underlying averaged alpha into account which assumes that once alpha is blocked, it becomes desynchronized and is reset once it reappears; the other took averaged alpha into account, used SN-N data, and assumes that alpha is ever-present but blanketed during photic stimulation and does not appear to be reset when it reappears.

Whether one or the other model is appropriate depended upon intensity in this study. The VER(SN) activity looked to be much the same as averaged alpha taken over the same interval of time when a low intensity flash was used; however, when a high intensity flash was used, the alpha activity was desynchronized, and phase appeared to be reset whenever alpha-like activity reoccurred.

References

Andersen, P. & Andersson, S. A. Physiological basis of the alpha rhythm. New York: Appleton-Century-Crofts, 1968.

Barlow, J. S. & Brazier, M. A. B. The pacing of EEG potentials of alpha frequency by low rates of repetitive flash in man. Electroencephalography and Clinical Neurophysiology, 1957, 9: 161-162.

Barlow, J. S. & Estrin , T. Comparative phase characteristics of induced and intrinsic alpha activity. Electroencephalography and Clinical Neurophysiology, 1971, 30: 1-9.

Barlow, J. S. & Lansing, R. W. Rhythmic afterwaves to flashes in relation to amplitude and phase of foregoing and subsequent background alpha activity (alpha blocking). Electroencephalography and Clinical Neurophysiology, 1969, 27: 695.

Bechtereva, N. P. & Zontov, V. V. The relationship between certain forms of potentials and the variations in brain excitability (based on EEG recorded during photic stimuli triggered by rhythmic brain potentials). Electroencephalography and Clinical Neurophysiology, 1962, 14: 320-330.

Bergamini, L. & Bergamasco, B. Cortical evoked potentials in man. Springfield, Ill.: Charles C. Thomas, 1967.

Bishop, G. H. Cyclic changes in excitability of the optic pathway of the rabbit. American Journal of Physiology, 1933, 103: 213-224.

Callaway, E. Factors influencing the relationship between alpha activity and visual reaction time. Electroencephalography and Clinical Neurophysiology, 1962, 14: 674-682.

Callaway, E. & Alexander, J. D. The temporal coding of sensory data: an investigation of two theories. Journal of General Psychology, 1960, 62: 293-309.

Callaway, E. & Layne, R. S. Interaction between the visual evoked response and two spontaneous biological rhythms: the EEG alpha cycle and the cardiac arousal cycle. Annals of New York Academy of Science, 1964, 112: 421-431.

Callaway, E. & Yeager, C. L. Relationship between reaction time and electroencephalographic alpha phase. Science, 1960, 132: 1765-1766.

Canter, M. B. & Fleming, D. E. Cortical potentials evoked in man by brief photic stimulus trains. Perceptual-electrophysiological correlates. Vision Research, 1966, 6: 199-206.

Chapman, R. M., Shelburne, S. A., & Bragdon, H. R. EEG alpha activity influenced by visual input and not by eye position. Electroencephalography and Clinical Neurophysiology, 1970, 28: 183-189.

Cigánek, L. The EEG response (evoked potential) to light stimulus in man. Electroencephalography and Clinical Neurophysiology, 1961, 13: 165-172.

Cigánek, L. Excitability cycle of the visual cortex in man. Annals of New York Academy of Science, 1964, 112: 241-253.

Cohn, R. Rhythmic after-activity in visual evoked responses. Annals of New York Academy of Science, 1964, 112: 281-291.

Creutzfeldt, D. C. & Kuhnt, U. The visual evoked potential: physiological, developmental and clinical aspects. In W. Cobb and C. Morocutti (Eds.), The evoked potentials: Electroencephalography and Clinical Neurophysiology, Suppl. 26., Amsterdam: Elsevier, 1967. Pp. 29-41.

Donchin, E. & Lindsley, D. B. Averaged evoked potentials and reaction times to visual stimuli. Electroencephalography and Clinical Neurophysiology, 1966, 20: 217-223.

Dustman, R. E. & Beck, E. C. Phase of alpha brain waves, reaction time and visually evoked potentials. Electroencephalography and Clinical Neurophysiology, 1965, 18: 433-440.

Eason, R. G., Aiken, L. R., White, C. R., & Lichtenstein, M. Activation and behavior: II Visually evoked cortical potentials in man as indicants of activation level. Perceptual and motor skills, 1964, 19: 875-895, Monograph Suppl. 3-V19.

Eason, R. G., Harter, M. R., & White, C. T. Effects of attention and arousal on visually evoked cortical potentials and RT. Physiology and Behavior, 1968, 4: 283-289.

Gaarder, K. Averaged brain activity following saccadic eye movement. Science, 1964, 146: 1481-1483.

Gaarder, K., Koresko, R., & Kropel, W. The phasic relation of a component of alpha rhythm to fixation saccadic eye movements. Electroencephalography and Clinical Neurophysiology, 1966, 21: 544-551.

Gastaut, H., Regis, H., Lyagoubi, S., Mano, T., & Simon, L. Comparison of the potentials recorded from the occipital, temporal and central regions of the human scalp, evoked by visual, auditory and somato-sensory stimuli. In W. Cobb and C. Morocutti (Eds.), The evoked potentials: Electroencephalography and Clinical Neurophysiology, Suppl. 26, Amsterdam: Elsevier, 1967. Pp. 19-29.

Goldstein, S. Phase coherence of the alpha rhythm during photic blocking. Electroencephalography and Clinical Neurophysiology, 1970, 29: 127-136.

Harter, M. R. Excitability cycles and cortical scanning: A review of two hypotheses of central intermittency in perception. Psychological Bulletin, 1967, 68, No. 1: 47-58.

Harter, M. R. & White, C. T. Perceived number and evoked cortical potentials. Science, 1967, 156, No. 3773: 406-408.

Harter, M. R. & White, C. T. Effects of contour sharpness and check-size on visually evoked cortical potentials. Vision Research, 1968, 8: 701-711.

Hays, W. L. Statistics. New York: Holt, Rinehart and Winston, 1963.

Horstfehr, H. O. The spatial sum of evoked responses to a single stimulus (light or sound) recorded from the human scalp. In W. Cobb and C. Morocutti (Eds.), The evoked potentials: Electroencephalography and Clinical Neurophysiology, Suppl. 26, Amsterdam: Elsevier, 1967. Pp. 139-146.

Joseph, J., Rémond, A., Rieger, H., & Lesèvre, N. The alpha average. II Quantitative study and the proposition of a theoretical model. Electroencephalography and Clinical Neurophysiology, 1969, 26: 350-360.

Katzman, R. The validity of the evoked response in man. Annals of New York Academy of Science, 1964, 112: 238-240.

Kooi, K. A. & Bagchi, B. K. Observations on early components of the visual evoked response and occipital rhythm. Electroencephalography and Clinical Neurophysiology, 1964, 17: 638-643. (a)

Kooi, K. S. & Bagchi, B. K. Visual evoked responses in man: Normative data. Annals of New York Academy of Science, 1964, 112: 254-269. (b)

Lansing, R. W. Relation of brain and tremor rhythms to visual reaction time. Electroencephalography and Clinical Neurophysiology, 1957, 9: 497-504.

Lansing, R. W., Schwartz, E., Lindsley, D. B. Reaction time and EEG activation under alerted and nonalerted conditions. Journal of Experimental Psychology, 1959, 58: 1-7.

Latour, P. L. The eye and its timing. National Defence Research Council T.N.O., Soesterberg, The Netherlands, 1961, Pp. 1-16.

Latour, P. L. Evidence of internal clocks in the human operator. Acta Psychologica, 1967, 27: 341-348.

Lindsley, D. B. Psychological phenomena and the electroencephalogram. Electroencephalography and Clinical Neurophysiology, 1952, 4: 443-456.

Magnus, O. & Ponsen, L. The influence of the phase of the alpha rhythm on the cortical evoked response to photic stimulation. Electroencephalography and Clinical Neurophysiology, 1965, 18: 427-428.

Mulholland, R. & Evans, C. R. An unexpected artifact in the human electroencephalogram concerning the alpha rhythm and the orientation of the eyes. Nature, 1965, 207: 36-37.

Mulholland, R. & Evans, C. R. Oculomotor function and the alpha activation cycle. Nature, 1966, 211: 1278-1279.

Peacock, S. M. Averaged "after-activity" and the alpha regeneration cycle. Electroencephalography and Clinical Neurophysiology, 1970, 28: 287-295.

Rémond, A. & Lesèvre, N. Variations in average visual evoked potential as a function of the alpha rhythm phase ("auto-stimulation"). In W. Cobb and C. Morocutti (Eds.), The evoked potentials: Electroencephalography and Clinical Neurophysiology, Suppl. 26, Amsterdam: Elsevier, 1967. Pp. 42-52.

Rémond, A., Lesèvre, N., Joseph, J., Rieger, H., & Lairy, G. The alpha average. I Methodology and description. Electroencephalography and Clinical Neurophysiology, 1969, 26: 245-265.

Rodin, E. A., Grisell, J. L., Gudoba, R. D., & Zachary, G. Relationship of EEG background rhythms to photic evoked responses. Electroencephalography and Clinical Neurophysiology, 1965, 19: 301-304.

Spong, P., Haider, M., & Lindsley, D. B. Selective attentiveness and cortical evoked responses to visual and auditory stimuli. Science, 1965, 148: 395-397.

Walsh, E. G. Visual reaction time and the alpha rhythm, an investigation of the scanning hypothesis. Journal of Physiology, 1952, 118: 500-508.

White, C. T. Temporal numerosity and the psychological unit of duration. Psychological Monographs, 1963, 77 (12, Whole No. 575).

White, C. T. & Eason, R. G. Evoked cortical potentials in relation to certain aspects of visual perception. Psychological Monographs, 1966, 80 (24, Whole No. 632).

Wilkinson, R. T. Evoked response and reaction time. Acta Psychologica, 1967, 27: 235-245.

Wilkinson, R. T. & Morlock, H. C. Auditory evoked response and reaction time. Electroencephalography and Clinical Neurophysiology, 1967, 23: 50-56.

APPENDIX A

TABLE 1

VARIANCE ANALYSIS SUMMARY FOR VER (SN)

Source of Variation	df	MS	Error Term	F	W^2
A. Phase	4	1.5028	E+G	20.0107**	.02
B. Intensity	2	4.9040	F+G	95.2200**	.45
C. Replications	1	.2167	G	4.5621	.01
D. Phase X Intensity	8	.3813	G	8.0274**	.14
E. Phase X Replications	4	.1301	G	2.7389	.02
F. Intensity X Replications	2	.0675	G	1.4211	.02
G. Phase X Intensity X Replications	8	.0475			
Total	29				

* $p < .05$
** $p < .01$

TABLE 2

VARIANCE ANALYSIS SUMMARY FOR EARLY ALPHA BLOCKING (SN)

Source of Variation	df	MS	Error Term	F	W^2
A. Phase	4	.0397	E+G	1.3233	.05
B. Intensity	2	.1936	F+G	10.8764**	.28
C. Replications	1	.0403	G	1.9659	.02
D. Phase X Intensity	8	.0421	G	2.05	.13
E. Phase X Replications	4	.0489	G	2.3854	.09
F. Intensity X Replications	2	.0068	G		
G. Phase X Intensity X Replications	8	.0205			
Total	29				

* $p < .05$
** $p < .01$

TABLE 3

VARIANCE ANALYSIS SUMMARY FOR LATE ALPHA BLOCKING (SN)

Source of Variation	df	MS	Error Term	F	W^2
A. Phase	4	.0270	E+G	1.0112	.03
B. Intensity	2	.0580	F+G	3.0688	.12
C. Replications	1	.0864	G	4.0000	.08
D. Phase X Intensity	8	.0222	G	1.0278	.03
E. Phase X Replications	4	.0370	G	1.7130	.15
F. Intensity X Replications	2	.0082	G		
G. Phase X Intensity X Replications	8	.0216			
Total	29				

*$p < .05$
**$p < .01$

TABLE 4

VARIANCE ANALYSIS SUMMARY FOR REACTION-TIME LATENCY

Source of Variation	df	MS	Error Term	F	W^2
A. Phase	4	1011.6167	E+G	.8904	.01
B. Intensity	2	13851.5333	F+G	30.4788**	.53
C. Replications	1	2502.5333	G	2.2986	.03
D. Phase X Intensity	8	232.2667	G		
E. Phase X Replications	4	1231.1167	G		
F. Intensity X Replications	2	189.6334	G		
G. Phase X Intensity X Replications	8	1088.7167			
Total	29				

*$p < .05$
**$p < .01$

TABLE 5

VARIANCE ANALYSIS SUMMARY FOR REACTION-TIME VARIABILITY

Source of Variation	df	MS	Error Term	F	W^2
A. Phase	4	278.9667	E		
B. Intensity	2	152.2334	F		
C. Replications	1	529.2000	G	2.1602	.05
D. Phase X Intensity	8	182.9417	G		
E. Phase X Replications	4	859.7000	G	3.5093	.27
F. Intensity X Replications	2	395.1000	G		
G. Phase X Intensity X Replications	8	244.9750			
Total	29				

*$p < .05$
**$p < .01$

TABLE 6

VARIANCE ANALYSIS SUMMARY FOR VER (SN-N)

Source of Variation	df	MS	Error Term	F	W^2
A. Phase	4	.1686	E	6.4106*	.03
B. Intensity	2	4.6591	F, G	48.6844**	.72
C. Replications	1	.0010	G		
D. Phase X Intensity	8	.2066	G	2.6453	.09
E. Phase X Replications	4	.0263	G		
F. Intensity X Replications	2	.1661	G	2.1268	.02
G. Phase X Intensity X Replications	8	.0781			
Total	29				

*p < .05
**p < .01

TABLE 7

VARIANCE ANALYSIS SUMMARY FOR EARLY ALPHA BLOCKING (SN-N)

Source of Variation	df	MS	Error Term	F	W^2
A. Phase	4	.0072	E+G	.8571	.01
B. Intensity	2	.0357	F+G	4.3537*	.19
C. Replications	1	.0128	G	1.6623	.04
D. Phase X Intensity	8	.0103	G	1.3377	.09
E. Phase X Replications	4	.0097	G		
F. Intensity X Replications	2	.0026	G		
G. Phase X Intensity X Replications	8	.0077			
Total	29				

*$p < .05$
**$p < .01$

TABLE 8

VARIANCE ANALYSIS SUMMARY FOR LATE ALPHA BLOCKING (SN-N)

Source of Variation	df	MS	Error Term	F	W^2
A. Phase	4	.0050	E	.4587	.03
B. Intensity	2	.0304	F	7.6000	.14
C. Replications	1	.0124	G	.7470	.03
D. Phase X Intensity	8	.0157	G	.9458	.02
E. Phase X Replications	4	.0109	G		
F. Intensity X Replications	2	.0040	G		
G. Phase X Intensity X Replications	8	.0166			
Total	29				

*$p < .05$
**$p < .01$

APPENDIX B

Latin Square for Experimental Conditions

General

Subjects	Repl.	Reaction Time			No Reaction Time		
		L	M	H	L	M	H
1	1	1	2	3	4	5	6
	2	6	5	4	3	2	1
2	1	2	3	6	1	4	5
	2	5	4	1	6	3	2
3	1	3	6	2	5	1	4
	2	4	1	5	2	6	3

Alpha Phase Order						Subjects		
Oms	25ms	50ms	75ms	100ms	N/S	1	2	3
1	2	3	4	5	N/S	RT L	RT M	RT H
2	3	N/S	1	4	5	RT M	RT H	NRT L
3	N/S	2	5	1	4	RT H	NRT L	NRT M
4	5	1	2	N/S	3	NRT L	NRT M	NRT H
5	1	4	N/S	3	2	NRT M	NRT H	RT L
N/S	4	5	3	2	1	NRT H	RT L	RT M

INSTRUCTIONS TO EXPERIMENTER

I. CHECK inkwells, RT paper and Polygraph paper.

II. TURN ON EQUIPMENT

1. Master switch
2. Reaction time Power - On
3. Pull out 28v supply on Solid State
4. Turn on two Power Supplies
5. Turn on Grass PS-2 photostimulator -- 2 switches.
6. Turn on White noise POWER
7. Turn on Polygraph
8. Check oscilloscope and CAT to make sure they are on.

III. PREPARE SUBJECT

IV. ADJUST SCHMITT TRIGGER and record value for each S.

V. ADJUST DELAY on photostimulator so that in the Phase 1 position the stimulus will be presented at the trough between the 1st and 2nd alpha.

VI. POST SUBJECT'S SESSION SCHEDULE

VII. BEFORE STARTING EACH BLOCK

1. Advance RT tape and reset.
2. Reset mechanical and electrical timers.
3. Set Phase !
4. Erase and start CAT.
5. Warn the S that starting is imminent.
6. Turn on white noise.
7. Plug in trigger if in Alpha Phase or Clock if No Synch.

VIII. DURING EACH BLOCK

1. Always monitor scope -- observing phase and stimulus presentation.
2. Monitor CAT for stimulus presentation.
3. Monitor polygraph. Observe EEG and check to see if the S is responding properly.
4. Label Polygraph and RT with name, date, block, intensity, response.
5. Label graph paper and place into position.

IX. AT END OF EACH BLOCK

1. Unplug trigger or turn off clock.
2. Turn off polygraph.
3. Turn off white noise and talk to the S.
4. Print Out
    1. Invert RT in Channel 2.
    2. Put Channel 1 into 4.
    3. Subtract Channel 3 from 4.
    4. Print out.

PROCEDURE WITH SUBJECT

1. Put on glasses for dark adaptation for 30 minutes.

2. Place electrode in midline one inch above inion. Reference electrode will be clipped to the right ear lobe. Be sure to put paste on electrode.

3. Check resistance -DC RX10,000.

4. Be sure that the electrodes are not entangled in the glasses so that glasses can be removed.

5. Place the S in chair. Ask the S to close eyes while Ganzfeld is being placed into position and to keep them closed until instructed otherwise which will be when E darkens the sound-and light-shielded room.

6. Adjust head rest so that the S is as comfortable as possible in an upright position and is approximately 80 cm. from visual stimulus display.

7. Plug in electrodes -- notched edge toward control room.

8. Instruct the S to stay as relaxed as possible and yet alert enough to make responses when appropriate.

9. Stress to the S that movement must be kept to a minimum during the actual recording period. If it is necessary to blink, or move head, suggest they try to do so right after a click has been heard which at times indicates when a trial has ended.

10. Explain to the S that his alpha activity will trigger the stimulus situation and that there will be no set time when stimulus will be presented. Stimuli will be a light flash which will be randomly presented 50% of the time; during the other 50% of the time a flash will not be presented.

11. There are two possible ways to respond to the stimulus condition. During any one session (day) the S will be asked to use only one method of responding and will be told at the beginning of the session what is expected.
    The S will make the same response with the left hand during every session. That is -- when he hears a click, it will indicate the end of a trial and will symbolize the question, "Did you see a light flash?". If the answer is "No," he will gently push the button.

The response is necessary to keep tabs on whether or not a stimulus was perceived.
The other response situation involves the use of a reaction-time key which the S is asked to release if he sees a light flash. If the key is released, the click will not be presented. If the key is held down, the click will sound and ask the usual question. In the RT condition, the answer will probably be "No"; otherwise a key release would have prevented the "questioning" click.

12. A Session will involve six blocks with 64 trials within each block. Each block will take approximately 5 mins. There will be a rest period between each session while E is printing out.

13. Tell the S at beginning of session of brief delay for adjusting equipment.

INSTRUCTIONS TO SUBJECT

This study is looking into the relationship between alpha-type brain activity and perceived visual light flashes. For this reason, we would like you to remain as relaxed as possible and yet alert enough to perceive the light flash if presented. Please keep your eyes open during trial runs.

During this session there will be six trial runs. Each trial run will take about 4-1/2 mins. During each run, 32 light flashes will be presented randomly over 64 trial periods about 4 secs. long. Actually your alpha activity will determine the time when stimulus will be presented.

There will be a rest period between each trial run for about 2 mins. You may move, stretch, talk to me over the intercom, etc. I will warn you at the beginning of each run, and the white noise will indicate the actual beginning.

1. Today I want you to keep this reaction-time key down at all times unless you see a light flash. When you see a light flash, release the key as soon as possible. If you do not release the key, you will hear a click at the end of the trial which is asking the question, "Did you see a light?" If you did not, please gently push the button in your left hand. Be sure that you do not move your head when you do this. Any extra movement will show up on the record.

2. Today I want you to listen for the click which will come at the end

of each stimulus period. It is asking you the question, "Did you see a light flash?" If you did not, please gently push the button in your left hand. Remember that any extra movement will show up on the records.

Do you have any questions? If anything comes up during the Session, I will be able to hear you between the trial runs over the intercom.